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GUIDES FOR SHORT-TERM EXPOSURES OF THE PUBLIC TO AIR POLLUTANTS. VIII. GUIDE FOR CHLORINE

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Guides for Short-Term Exposures of the Public to Air Pollutants

VIII. Guide for Chlorine

by

The Committee on Toxicology

of the

National Academy of Sciences - National Research Council

Washington, D. C.

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The members of the committee selected to undertake this project and prepare this report were chosen for recognized scholarly competence and with due consideration for the balance of disciplines appropriate to the project. Responsibility for the detailed aspects of this report rests with that committee.

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INTRODUCTION

The Office of Air Programs of the Environmental Protection Agency has focused its initial concerns on long-term exposures of the public to air pollutants. In addition to the long-term levels there are occasional circumstances wherein the public may be exposed briefly to relatively high concentrations. For example, batch process techniques in industries may result in pulses of effluent. The testing and launching of rockets releases exhaust products. Rapidly changing meteorological conditions may result in short periods of locally high concentrations of stack effluents. Accidental releases of chemicals sometimes occur in industrial areas or during transport, and may lead to public exposure.

Recognizing that these occasional peak additions to the ambient exposures of the public do occur, the Office of Air Programs has requested the assistance of the Committee on Toxicology of the National Academy of Sciences - National Research Council in providing Guides for Short-Term Exposure Limits for Air Pollutants.

In preparing these guides, the Committee utilized the criteria described in the NAS-NRC document entitled "Basis for Establishing Short-Term Exposure limits of the Public to Atmospheric Pollutants" (29). Primary consideration was given to literature dealing with single or intermittent brief exposures to the contaminant in question, in this case chlorine.

CHLORINE

Chlorine is a greenish yellow gas with a molecular weight of 70.9, a boiling point of -34°C, and a specific gravity 2.47 times that of air. It is readily liquefied and is normally shipped in the liquid state in steel containers (cylinders, tank cars, etc.).

Liquid chlorine exerts a vapor pressure of 3.66 atmospheres at 0°C, 6.62 atmospheres at 20°C, 8.75 atmospheres at 30°C, and 41.7 atmospheres at 100°C (1-3).

Chlorine is slightly soluble in water, 2.26 liters of gas dissolving in one liter of water at 20°C (~ 0.1 moles/l). (For comparative purposes the solubility of ammonia, hydrogen chloride, and chlorine at 0°C is 53 moles/l, 23 moles/l, and 0.2 moles/l respectively.)

The odor threshold of chlorine has been reported to be about 5 ppm (15 mg/m^3) (1), 3.5 ppm (10.2 mg/m^3) (4), and 0.3 ppm (0.9 mg/m^3) (5) in this country. Russian scientists report odor threshold levels of 0.2 ppm (0.6 mg/m^3) to 0.4 ppm (1.2 mg/m^3) (6, 7).

Effects on Animals

Chlorine gas is classified as a primary irritant of the entire respiratory tract (3). Other irritating gases such as ammonia and hydrogen chloride which are more soluble than Cl_2 are trapped by the moisture on the lining of the nose and throat and irritate primarily those organs. Less soluble irritant gases such as phosgene pass through the upper respiratory tract to a greater extent and damage pulmonary tissue. Chlorine is capable of affecting both the upper respiratory passages and the deeper structures of the lung (2).

An accepted theory explains the action of chlorine on the basis of oxidation. According to this theory chlorine obtains hydrogen from the water of moist tissue, causing the formation of nascent oxygen and hydrogen chloride. The oxygen produces major damage, which is made worse by the HCl. The result is the tissue irritation and/or damage observed upon excessive exposure (3).

Exposure for 1/2 to 3/4 hour to air containing 1,000 ppm Cl_2 kills even large animals almost immediately. If the animals survive for a longer period, death ensues from pulmonary edema. Respiratory rate increases in air containing from 200 to 1,000 ppm, but slows at concentrations above 10,000 ppm until respiratory arrest occurs (1).

Cralley (8) examined the effects of chlorine on mucociliary activity of sections of excised rabbit trachea. The excised tracheal tissue was maintained in a constant temperature-humidity chamber and observed microscopically. Thirty ppm of Cl_2 for 5 minutes and 18-20 ppm for 10 minutes caused cessation of ciliary activity without recovery. Cralley reports that the concentration necessary to produce ciliostasis is of the same order as that which produces immediate irritation of the throat, i.e., 15 ppm. Concentrations ranging from 200 ppm for less than one minute to 20 ppm for about 2-1/2 minutes caused reversible ciliostasis.

Rats susceptible to spontaneous pulmonary disease (SPD) and specific-pathogen-free (SPF) rats were exposed to chlorine in order to examine the differences in response (26, 27). Ten-week-old SPD animals exposed to 14-18 ppm for one hour daily for a total of 24 hours exhibited little change while alive; on sacrifice the day after the last exposure, ulcerative inflammatory lesions in the trachea and main bronchi were apparent. The animal sacrificed one week later exhibited no abnormalities. Following exposure to 30 ppm Cl_2 for a total of 50 hours, the rats showed marked goblet cell proliferation; about one half of the exposed group died during exposure (26).

Exposure of adult SPD rats (18-31 weeks old) to 40-80 ppm for 2 hours per day for up to 5 weeks killed about 40% of the animals. There was necrosis of the entire mucosa of the main bronchus and less severe damage in the peripheral bronchi. Survivors sacrificed after the last exposure showed no differences when compared to their controls (26).

Young (14 weeks) SPF animals exposed at 40 ppm for a total of 43 hours exhibited only minimal goblet cell proliferation and none of the exposed animals died (27).

Twenty-week old female SPF and SPD rats were exposed to Cl_2 at 70 and 118 ppm for three hours per day for a total of 17 hours. Male animals 10-14 weeks old were exposed to increasing concentrations of Cl_2 (from 34 to 170 ppm) for three hours per day for a total of 60 hours and a mean Cl_2 concentration of 90 ppm. After exposure the SPD animals showed a more marked mucus secretion and goblet cell proliferation than did the SPF animals. The latter had no polymorphonuclear (PMN) reaction in the lungs, while the SPD animals exhibited an intense PMN reaction (27).

The exposure of cats to a concentration of 300 ppm for one hour caused death after a period during which the conjunctivae were inflamed and there was coughing and dyspnea (1).

Effects on Humans

The sensitivity of persons to chlorine varies greatly. Although they may work without interruption in air containing 1 or 2 ppm (3-6 mg/m^3), 3-6 ppm (9-18 mg/m^3) may cause a stinging or burning sensation in the eyes, nose, or throat, and sometimes headache. Exposure at this concentration may produce redness or watering of the eyes, sneezing, coughing and huskiness, or loss of the voice. Exposure for one half to one hour to 14-21 ppm (41-61 mg/m^3) is dangerous, and 100 ppm (290 mg/m^3) is intolerable after one minute (1).

Persons not familiar with the odor or effects of Cl_2 may continue to expose themselves at levels as high as 5 to 8 ppm for periods sufficiently long to cause the results that are normally associated with a mild acute exposure (28).

Zielhuis (9) derived the following dose-response relationship based on a review of the literature regarding the effects of Cl_2:

> "At 1-3 ppm (3-9 mg/m^3) there may be slight irritation; 4 ppm (12 mg/m^3) cannot be exceeded for more than one hour without discomfort; above 5 ppm (15 mg/m^3) there is severe irritation of the eyes, nose, and respiratory tract which becomes intolerable for more than a few minutes; more than 30 ppm is dangerous and possibly lethal after 30 minutes. This information suggests that the concentration should be kept below 10 ppm (29 mg/m^3) to prevent pulmonary oedema while for exposure to 30-60 minutes the concentrations should be low enough to prevent disabling discomfort."

A transient bronchospasm will occur in almost every exposure to Cl_2 but after minor exposures it is detectable only in ventilation studies performed soon after exposure. After more severe exposure, it may be detectable for several days. Individuals with decreased pulmonary or cardiac reserve may suffer more severely than healthy individuals. The very young and the very old may also show a greater response to Cl_2 exposure (28).

Kowitz et al. (10) studied the effects of Cl_2 on men accidentally exposed in a transportation accident. Of approximately 150 men exposed, 59 were included in the analysis: Decreased vital capacity, increased elastic work of breathing, and a decreased diffusing capacity resulting from the exposure in the 59 subjects were seen when they were evaluated between two and three years after the incident. Eleven who were hospitalized were evaluated with respiratory-function tests on

four occasions during the following two to three years. The mean acute respiratory-function changes of the 11 subjects were compatible with an alveolocapillary injury. This was followed by repair over the following 2-3 year period. Increased airway resistance, and a decrease in lung volume and diffusing capacity remained at the end of the study period and were considered to result from the exposure.

Kaufman and Burkons (11) studied 18 subjects accidentally exposed to Cl_2. The subjects were examined during the initial 24 to 48 hours and again 1, 2, 4, 12, and 14 months after the Cl_2 exposure. Physiological tests included vital capacity, forced expiratory volume, residual volume, airway resistance, diffusing capacity, and arterial oxygen tension. X-rays were taken during these examinations. From the results the authors conclude that evidence of obstructive airway disease was observed within hours following a single but rather severe chlorine gas exposure. The physiologic disturbance was not evident 30 to 90 days following the exposure. Clinical signs and symptoms as well as roentgenograms returned to normal within 30 days and remained so. Two of the subjects showed severe pulmonary edema after exposure and were cyanotic for several days but showed no permanent sequelae 14 months later.

Weill et al. (35) studied 12 people 7 years after an accidental chlorine exposure. They found no significant changes in pulmonary function tests or on chest X-ray films.

In high concentrations Cl_2 irritates the skin, causing burning and irritation with the possibility of blister formation (1). However, such irritations would be expected to occur only when exposure of the more sensitive eyes and pulmonary system is prevented by the use of respiratory protective devices (3).

Table I lists various concentrations of Cl_2 along with findings or remarks concerning these concentrations.

TABLE I

Concentration	Remarks	Ref.
0.3 - 3.5 ppm	Range of reported odor thresholds	
1 ppm	TLV, OSHA time-weighted average	16, 25
1 - 2 ppm	Men can work without interruption	1
1 - 3 ppm	There may be slight irritation	9
3 ppm	Pennsylvania short-term limit for 5 min	19
3 ppm	Permissible excursion for 15 min based on TLV	16
3 ppm	Recommended as 60 min EEL*	9
3 - 6 ppm	Stinging or burning in the eyes, nose, and throat, and perhaps headache due to sinus irritation. Maybe watering of eyes, sneezing, coughing, and bloody nose and/or blood-tinged sputum.	1
4 ppm	Recommended 30 min EEL*	9
5 ppm	Recommended 15 min EEL*	9
Above 5 ppm	Severe irritation of the eyes, nose, and respiratory tract, which becomes intolerable after a few minutes.	9
7 ppm	Recommended 5 min EEL*	9
14 - 21 ppm	Dangerous within 1/2 to 1 hr	1
34 - 51 ppm	Lethal in 1 - 1-1/2 hr	23

* This "EEL" is for an emergency in a manufacturing area and its neighborhoods, storage facilities and the surrounding areas, and during transport of the chemical, suggested by Zielhuis in 1970.

Effects on Materials

Chlorine has a very strong chemical affinity for many substances. It will react with almost all the elements and with many organic and inorganic compounds. At elevated temperatures it reacts vigorously with most metals. At ordinary temperatures, dry, liquid, or gaseous Cl_2 does not corrode steel. In the presence of moisture, highly corrosive conditions exist due to the formation of hydrochloric acid and hypochlorous acid (12).

Under the conditions recommended in the STPL's and PEL's, no significant effects on materials would be expected. However, continuous exposure at these levels could enhance corrosion of some materials normally attacked by Cl_2.

Murphy et al. (24) determined the effect of gaseous Cl_2 flowing through an orifice in grade 70 steel under water. The experiment was performed to study the release characteristic of leaking submerged Cl_2 cylinders. They reported that Cl_2 at 10 psig flowing through a hole 0.030 inch in diameter and 0.625 inch long had, as a function of time, a constant mass flow rate of 2 lb/hr for three hours and then an abrupt four-fold increase in flow rate to 7.5 lb/hr. One hour later a second increase in flow was observed. A similar phenomenon was observed with gaseous Cl_2 under 90 psig except that the initial flow rate was greater (about 3.4 lb/hr, increasing to 72 lb/hr) and the time interval between flow changes was reduced. Examination of the hole after each experiment indicated that the original opening was enlarged, by corrosion of the steel, from the outside; the opening had the form of a truncated cone expanding outward.

Further information concerning the effects of Cl_2 on specific materials may be found in engineering references such as Metals Handbook (22).

Effects on Plants

Chlorine is capable of causing marginal and tip necrosis in plants; younger leaves being less susceptible than middle-aged or older leaves (13).

Brennan et al.(14) exposed a variety of plants to Cl_2 at concentrations ranging from 0.1 to 1.5 ppm for two to four hours. They report that the most common effects were bleaching of the foliage at low levels and necrosis at somewhat higher levels.

Radish and alfalfa were the two most sensitive species tested, showing injury at 0.1 ppm for two hours. Tobacco, zinnia, gomphrena, onion, corn, mustard, and sunflower were damaged by 0.1 to 0.25 ppm for four hours. Tomato, cucumber, dahlia, nasturtium, petunia, pinto bean, squash, corn-pea, geranium, and azalea were damaged at levels of 0.5 to 0.8 ppm for four hours. Polygonum, oxalis, and pepper were uninjured at levels of 1.5 ppm Cl_2 for three hours.

Chloride accumulation in the exposed plants exhibited no correlation with the degree of Cl_2 exposure, nor was the damage to wet leaf surfaces significantly different from that to dry leaf surfaces.

Benedict and Breen (15) exposed a variety of weeds to Cl_2 at concentrations ranging from 0.5 to 2.5 ppm for four hours and reported that mustard, chickweed, and sunflower were the most susceptible weeds to Cl_2, while Kentucky bluegrass, Lamb's-quarters, and pigweed were the most resistant. Weeds grown under conditions of low soil moisture were reported to be more resistant to Cl_2 injury than plants grown under conditions of high soil moisture.

Analytical Methods

Several methods of analysis for chlorine in air have been described in the literature (30-32). A commonly used method of analysis and one recommended by the American Industrial Hygiene Association for air sampling relies on the production of a yellow color when Cl_2 reacts with ortho-tolidine (30). An air sample is collected in dilute sodium hydroxide, which is later acidified before the ortho-tolidine is added.

The Intersociety Committee for Ambient Air Sampling and Analysis (33) has published a method for the determination of Cl_2 in air. This method, involving the use of an acidic solution of methyl orange, is claimed to be adequate for many situations although it is not free of interferences.

Stahl (34) discusses and summarizes several other methods for the analysis of Cl_2 in the atmosphere.

No common single method for the determination of Cl_2 is free from interference by other oxidizing, reducing, and/or chloride-containing agents.

Short-Term Public Exposure Limits (STPL's)

Short-Term Exposures are those occurring at predictable

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Short-Term Public Exposure Limits (STPL's)

Short-Term Exposures are those occurring at predictable

times and arising from single or, occasionally, repeated events. Where exposure can be predicted there is no justification for submitting the public to any appreciable risk.

Experimental data as well as data collected from occupational situations indicate that the initial effect of chlorine above the odor threshold is irritation of the eyes and mucous membranes of the nasopharynx.

It is desirable to keep the short-term public limits below the level of irritation that may lead to significant discomfort. None of the following are considered to present any health hazard; they are based upon the probability of producing only mild odor and minimum irritation. For the purposes of the STPL's the Panel on Chlorine assumed that the frequency of exposure would be no more than once per month in order to prevent possible cumulative damage due to repeated exposure.

STPL's

10 min	1.0 ppm (2.9 mg/m^3)
30 min	0.5 ppm (1.4 mg/m^3)
60 min	0.5 ppm (1.4 mg/m^3)

The STPL's are to be considered Time-Weighted Averages, with a maximum excursion factor of 3 for the ten-minute level, and 2 for the 30- and 60-minute levels. Any excursions above the STPL should be compensated for by an equivalent reduced exposure during that time period.

Public Emergency Limits (PEL's)

While under optimum conditions the STPL's require that there be no adverse health effects, the PEL's differ from the STPL's by recognizing the possibility of some temporary discomfort provided that the effect is reversible and that no serious sequelae result from it. The following levels are recommended as PEL's for chlorine. It is thought that effects will be confined to strong odor and irritation of the mucous membranes.

PEL's

10 min	3 ppm (8.7 mg/m^3)
30 min	2 ppm (5.8 mg/m^3)
60 min	2 ppm (5.8 mg/m^3)

The PEL's are Ceiling Limits and are not to be exceeded.

These limits (STPL's and PEL's) are based on data primarily from industrial exposure of healthy people. There is reason to believe that persons having pre-existing pulmonary disease may be more susceptible to the effects of Cl_2, but data are not adequate to set levels for such persons. Therefore, the above limits are proposed as tentative values. The Panel recommends that appropriate research be done to determine the effects of Cl_2 on those likely to be more than normally susceptible to Cl_2.

The limits are recommended on the basis of human response. They were not set for the protection of vegetation or materials.

APPENDIX I

Established Limits:

TLV: 1 ppm (3 mg/m^3) (15-min excursion : 3 ppm) (16)

Russian Maximum Allowable Concentration (industrial):
0.001 mg/l (1 mg/m^3) (17)

Recommended EEL for submarine crews: 3 ppm for 60 min (18)

Pennsylvania Short-Term Limits: 3 ppm for 5 min (19)

Maximum allowable concentration in populated areas:
(Russian figures) (20)

average 24-hr concentration: 0.03 mg/m^3
maximal single concentration: 0.1 mg/m^3

Recommended EEL's for exposure: (9)

60 min	:	3 ppm
30 min	:	4 ppm
15 min	:	5 ppm
5 min	:	7 ppm

International MAC's in the workplace: (21)

German Federal Republic: 2 mg/m^3[1968]
German Democratic Republic: 1 mg/m^3[1963]
Great Britain: 3 mg/m^3[1955]
Hungary: 1 mg/m^3 [1956]
Poland: 1 mg/m^3 [1959]
Yugoslavia: 3 mg/m^3 [1957]
Czechoslovakia: mean MAC: 3 mg/m^3
peak MAC: 6 mg/m^3 [1969]

Department of Labor, Occupational Safety and Health Administration: 1 ppm (3.0 mg/m^3) (25)

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